Exhibit 24

of dopamine, but the "threshold" for clinical signs to be manifest varies depending on a variety of factors.

- *Exposure to pesticides*: Paraquat, rotenone and diazinon are recognized causes of Parkinson's disease. Recent research has shown there are at least 10 pesticides that are toxic to dopaminergic neurons involved in Parkinson's disease.[21] Pesticides are thought to damage dopaminergic neurons via the same route as MPTP and solvents i.e. oxidative stress and mitochondrial toxicity. Oxidative stress is an imbalance between the production of harmful free radicals (reactive oxygen species) and the body's ability to counteract them with antioxidants, leading to damage to brain cells. Mitochondria are membrane-bound cell organelles that generate most of the chemical energy needed to power the cell's biochemical reactions.
- *Heavy metals*: Heavy metals, such as iron (Fe), mercury (Hg), manganese (Mn), copper (Cu), and lead (Pb), have been linked to PD and contribute to its progression. [22] The mechanism of cell death with heavy metals is also oxidative stress.
- *Cleaning chemicals and solvents*: A growing body of literature has demonstrated that exposure to trichloroethylene (TCE) is a risk for developing Parkinson's disease. Epidemiologic data has become more compelling over the years. The first case report of PD developing in the setting of TCE exposure (through a work exposure) was published in 1999.[24] In 2008, a cluster of TCE exposed workers had a higher incidence of Parkinson's disease than the general population. This study demonstrated that TCE was toxic to mitochondria with a mechanism similar to MPTP. [25] A twin study authored by Dr. S. Goldman published in 2011 showed that exposure to TCE increased the risk of PD over 6 fold.[26] Twin studies are particularly relevant since twins share nearly identical genetic markers but differ only in their exposure to TCE. Two studies examined mortality among Marine and Navy personnel and among civilian employees at Camp Lejeune, as compared to those at Camp Pendleton.[27,28] This is the first in a series of studies that compare Camp Lejeune (where TCE levels were well above the allowable level) and Camp Pendleton (where there is no evidence to TCE contamination). Mortality hazard ratios at Camp Lejeune were significantly higher for many causes, including PD. The study estimated levels of TCE and PCE in water in the Hadnot Point system during the period 1975-1985 of 359 µg/L and 16 µg/L, respectively.[27,28] The Maximum Contaminant Level (MCL) set by the EPA is 5 µg/L for TCE and PCE.[84,85]

  The next major study looking specifically at those that served at Camp Lejeune between 1975 and 1985 was published in 2023.[29] This study evaluated records of more than 150,000 veterans that served either at Camp Lejeune or Camp Pendleton between 1997-2021. The levels of TCE at Camp Lejeune were modeled to be 70 times the allowable levels (deemed by the EPA) during that time frame. Results showed that those who served at least 3 months at Camp Lejeune had a 70% greater chance of developing Parkinson's disease than those that served at Camp Pendleton. This result was highly significant ($P<.001$). Camp Lejeune veterans also had increased symptoms of prodromal parkinsonism suggesting that they were more likely to develop Parkinson's disease over time. Follow up of this cohort of veterans from Camp Lejeune showed that those affected by Parkinson's disease progressed more quickly with shorter time to psychosis, fracture and falls.[30] These findings suggest the PD caused by TCE may progress more rapidly.

  A large study from 2024 which was a follow-up to two previous studies by Bove et al. aimed at evaluating mortality of Marines, Navy personnel and civilian workers at Camp Lejeune and at Camp Pendleton. The former were exposed to TCE and to PCE over a thirty-year period (1953-1985). The findings indicate a two-fold increase of mortality due to PD in

Marines and Navy personnel at Camp Lejeune, compared to Camp Pendleton.[31] In some of my own research, we identified a cluster of attorneys with Parkinson's disease and prodromal parkinsonism who were exposed to high levels of TCE from a contaminated site near their office.[7] Their rate of PD and prodromal parkinsonism is higher than expected for age and also higher than the control cohort. A recent series of case reports from Dorsey et al included a professional athlete who spent his early childhood at Camp Lejeune and developed PD at the age of 34.[32] The above referenced studies have shown that Parkinson's disease can manifest 40-50 years after exposure to TCE but the timing of disease onset is individually variable.

In addition to epidemiologic data, animal studies have consistently shown that exposure to TCE causes damage to the dopaminergic system which mimics the changes seen in PD. The toxicity of TCE was documented in rodents of both sexes via oral, intraperitoneal or inhalation routes of administration.[33,34] These effects are both dose and time dependent meaning the higher or longer the exposure to TCE, the greater the damage. In fact, studies show that inhalation of TCE is more toxic than ingestion due to greater dopaminergic degradation.[35] Any ingestion exposure is also associated with inhalation exposure. Activities such as showering or swimming in water contaminated with TCE would add to ingestion exposure via the inhalation pathway.

Animal studies show TCE results in damage to mitochondrial function, loss of dopamine containing neurons in the substantia nigra (which is the region affected in PD), increased inflammation, accumulation of alpha-synuclein, and increased activity of LRRK2 kinase, the most common mutation associated with familial PD. These findings strongly support human studies that TCE can induce brain damage consistent with that observed in Parkinson's disease. Potential mechanisms of action of TCE induced PD are related to TCE metabolites and gut microbiome changes. TaClo is a TCE metabolite which is structurally similar to MPTP and causes similar damage to dopamine containing neurons in rodents.[36] TaClo also stimulates LRRK2 kinase activity which as noted above is the most common genetic mutation associated with PD. TCE has been shown to change the gut microbiome in the rodent model of TCE exposure which mimics the changes in gut microbiome changes in humans with PD.[37] Changes in gut microbiome are thought to alter the gut-brain axis seen in PD.

D. Dose level of TCE exposure at Camp Lejeune

TCE monthly exposure in the water supply at Camp Lejeune between 1975-1985 was modeled to reflect a mean monthly average level of be 366 ug/l-M.[29] The Maximum Contaminant Level (MCL) set by the EPA is 5 micrograms/liter. From my review of the ATSDR water modeling data, Marines, civilians and their families who lived and or worked at the base during the approximately 3 decades of TCE contamination would have potentially been exposed to levels of TCE that far exceeded the regulatory levels set by the EPA. More importantly, based on the previously cited literature, it has been demonstrated clearly that exposure to Camp Lejeune's water supply for 3 months or more during the decade beginning in 1975, resulted in a 70% increase in the risk of developing Parkinson's disease.[29]

In 2017, the Agency for Toxic Substances and Disease Registry (part of the Federal government) concluded that there was "equipoise and above evidence" for causation for TCE and Parkinson's disease. Since then, epidemiologic data has made this argument even stronger. More recently, in December of 2024, the EPA banned all use of TCE and greatly restricted to use of PCE (perchloroethylene) based in part on the deleterious effects these chemicals have on the neurologic system.[49] The EPA's recent action is consistent with what has become increasingly known and recognized in the medical and scientific fields. We have recognized the causal association between TCE and PCE and Parkinson's Disease for several years., Published scientific literature proves this causal relationship. This is further explained in the reports of other experts in this case.

		Chart 1: 1L	Chart 2: ATSDR marine in training	Chart 3: Deposition informed activity days and ATSDR 6L & 3L exposures
	Cumulative ug/l-M	Cumulative consumption (total ug= days*concentration per L)	Cumulative consumption (total ug= days*concentration per ATSDR exposure assumptions)	Cumulative consumption (total ug= days*concentration per deposition exposure assumptions)
Hadnot Point				
TCE	3,195	90,063	390,333	281,649
PCE	65	1,830	7,931	5,723
VC	135	3,806	16,495	11,902
BZ	40	1,121	4,858	3,506

Exposure to amounts of TCE of 281,649 micrograms is clearly a substantial exposure. The compounding part for Mr. Sparks is that in addition to this very significant and substantial ingestion exposure, Mr. Sparks was also exposed to 5,723 of ug (micrograms) of PCE. TCE is a chemical that is causally related to Parkinson's disease. Mr. Sparks' TCE exposure put him at dangerously higher risk for the development of Parkinson's disease.

As with many diseases associated with toxin exposure, manifestation of symptoms comes years after the exposure. Mr. Sparks had an approximate 15-month exposure to toxic levels of TCE while serving at Camp Lejeune between March 25, 1974 and May 30, 1975.[86] In fact, his work area was adjacent to the most highly contaminated water supply on the base (Hadnot Point). Epidemiologic studies outlined above have shown a statistically significant association between toxic TCE exposure and Parkinson's disease. This is further supported by extensive animal research that documents TCE exposure causing the same pathology and mechanism of cell death that occurs in humans with PD Using the Bradford Hill framework applied by the general causation experts like Dr. Cannon, Dr. De Miranda, Dr. Miller, Dr. Costa and Dr. Boehme (strength of association, consistency, temporality, biologic gradient, plausibility, coherence experimental evidence and analogy), the overwhelming evidence strongly supports a causal relationship between TCE exposure and development of PD.

VI. Opinion on Causation

Based on my education, training, and expertise as a neurologist and a movement disorder specialist, and to a reasonable degree of medical certainty, based on the standard of causation under the Camp Lejeune Justice Act defined as "at least as likely as not," I conclude that Mr. Sparks Parkinson's disease is more likely than not, which exceeds the standard of is "at least as likely as not", due to his exposure to the water at Camp Lejeune containing TCE from March 25, 1974 to May 30, 1975.[86] [MOU1] [hs2]

A. Future Care Considerations

[redacted]